AFFIDAVIT OF BAXTER D. MONTGOMERY, MD

STATE OF TEXAS)
) ss.
COUNTY OF HARRIS)

BAXTER DELWORTH MONTGOMERY, MD, declares under penalty of perjury pursuant to Texas Civil Practice and Remedies Code Title 6 Section 132 that the foregoing is true and correct:

1. I am above the age of 18 and am competent to make this affidavit.
2. I am a Diplomate of the American Board of Internal Medicine, for cardiovascular diseases, licensed with the Texas State Board of Medical Examiners since 1991 under Permit Number H9549.
3. I am President and CEO of Houston Associates of Cardiovascular Medicine, PA. performing various forms of cardiovascular clinical care.
4. I have medical privileges at and serve as an attending physician for Memorial Hermann Hospital - The Texas Medical Center, The Heart and Vascular Institute at the Memorial Hermann Hospital - The Texas Medical Center,
5. I have chaired the Patient Safety Committee at Twelve Oaks Medical Center.
6. For 25 years until the present, I have served as Teaching Faculty for Cardiology Fellows at The Heart and Vascular Institute Memorial Hermann Hospital - The Texas Medical Center. (See my Curriculum Vita attached as **Exhibit A**).
7. Because cardiovascular disease has been the #1 cause of death in the United States, fifteen (15) years ago I began implementing lifestyle interventions within my clinical practice.

8. There are numerous peer reviewed studies on the benefits of a plant-based diet and lifestyle interventions in fighting disease.[1]

9. Currently, as President and CEO of Houston Associates of Cardiovascular Medicine, PA, I am responsible, with my staff, for the oversight and compliance with state and federal workplace and patient safety laws applicable to all healthcare facilities.

10. Therefore, I have general knowledge and working experience with the standards, regulations and guidance provided by the Department of Labor, Occupational Safety and Health Administration (OSHA). As part of my day-to-day duties as a healthcare clinical practitioner and compliance administrator during this Covid Pandemic, I constantly worked to ensure that my healthcare facility complies with patient and employee workplace safety standards.

11. Since March 2020 when the Pandemic was declared, I have treated many patients who have either tested positive for the virus that causes Covid-19, or have had Covid-19 related symptoms and I make this affidavit based on my clinical patient experience as well as based on my knowledge and experience as a practicing physician.

12. I have been retained by Attorney Jo Saint-George and Attorney Donna Este-Green of the non-profit organization the Women of Color for Equal Justice to give expert opinions based on my knowledge and experience as a licensed medical professional.

13. Specifically, I have been retained to provide opinions regarding whether or not employees who work in a healthcare setting with or without direct patient care responsibilities, or who work for municipal or private employer entities with or without direct public contact or have minimal public contact should be terminated by an employer for refusing to submit to the FDA emergency authorized injection called the "Covid-19 vaccine" based on applicable healthcare and general workplace safety standards as it relates to the medical efficacy of the COVID-19 vaccines and their potential risks.

[1] See Plant-based Research Database - https://plantbasedresearch.org/

14. In preparation of providing my opinions herein, I have reviewed the following: 1) New York City Department of Health and Mental Hygiene vaccine orders from August 10, 2021 to December 13, 2021, 2) applicable regulations of the U.S. Department of Labor, Occupational Safety and Health Administration, and 3) the affidavit and documents provided by Certified Industrial Hygienist, Mr. Bruce Miller, MS, CIH, President of Health & Safety, LLC.

BACKGROUND & PRELIMINARY OPINIONS

15. Between August 10, 2021 and December 13, 2021, the New York City Department of Health and Mental Hygiene (NYCDOHMH) issued approximate twelve (12) Covid-19 Emergency Orders applicable to New York City employees within its various agencies ("NYC Emergency Orders").[2]

16. Based on my review of the NYC Emergency Orders, the primary purpose of the orders was to mandate all New York City employee to submit to taking Covid-19 vaccinations as a workplace safety and health standard that reduces the spread and contraction of the virus that causes the communicable disease "Covid-19" in New York City facilities.

17. While the Covid Emergency Orders state that the Covid-19 vaccine requirements are for the benefit of the "health, safety, and welfare" of New York City residents, the orders only apply to New York City employees and do not indicate that there is a direct impact on the residents of the City. Based on my general public health knowledge as a clinician, the Emergency Orders are directed at City Employees in their workplace.

[2] See List of New York City Department of Health & Mental Hygiene list of Orders at https://www1.nyc.gov/site/doh/about/hearings-and-notices/official-notices.page

OPINIONS REGARDING COVID-19 WORKPLACE SAFETY REQUIREMENTS

18. My opinions regarding workplace safety requirements in general and for healthcare facilities are as follow and are made to a degree of medical certainty:

    a. the Covid-19 vaccines utilized in the United States are pharmacological medical treatments used to reduce symptoms that result from an infection of the viral pathogen and/or various variants of the Sars Cov2 virus, which causes the infectious disease identified by the Centers for Disease Control as Covid-19.

    b. "Covid-19 vaccines" do not eliminate the virus that causes infections of Covid-19 from the atmosphere of any in door facility. The virus that causes Covid-19 and/or its variants is an atmospheric contaminant or airborne hazard that should be controlled in any in-door facility which could stop or prevent the contraction of any infectious communicable diseases that can cause serious injury or death.

    c. Based on my general clinical knowledge of workplace safety standards for healthcare facilities and general industry facilities, the OSHA Standard at 29 C.F.R. § 1910.134 et seq.[3] titled "Respirator Protection" provides the minimum health and safety standard that any facility can utilize to reduce the risks of severe injury or death associated with any airborne contaminant that cannot be eliminate or controlled by other OSHA standards or methods.

    d. Because the Covid-19 vaccines cannot remove the virus that causes Covid-19 infections from the atmosphere of any facility, based on my clinical experience and hospital experience, N95 respirators or Powered Air Purification Respirators, which have the highest efficacy in reducing exposure to any airborne contaminate and can be used and are necessary, when nothing else eliminates the virus, to prevent the spread

of any airborne communicable disease according to the OSHA and CDC published guide titled "Hospital Respiratory Protection Program Toolkit – Resources for Respiratory Program Administrators" published in May 2015.[4]

e. There are entire industries of employees that are required to wear N95 respirators or PAPR's everyday eight hours a day, specifically industrial workers in the automotive, welding, commercial painting utilize this equipment to protect their employees from airborne contaminates. Therefore, employees in any workplace that have a risk of exposure to or can spread a viral airborne contaminant should be provided by an employer with at least an N95 respirator or a PAPR consistent with the OSHA standards set forth in 29 U.S.C. 1910.134, especially when necessary to protect the health of an employee as indicated in 1910.134(a)(2).

f. Based on my clinical experience treating patients with communicable disease, when the existing OSHA Respiratory Protection standards contained in Section 1910.134[5] are properly implemented in any facility, along with all other OSHA standards applicable to addressing communicable disease, vaccines, including the Covid-19 vaccine, (which cannot stop the spread or transmission of the virus) are not needed to provide a safe workplace for a employees.

g. While the OSHA standard 1910[6] titled Bloodborne pathogens recommends making Hep B vaccine available to employees who have occupational exposure to hepatitis B, the vaccine does not cure nor remove the blood-borne virus that can cause chronic infection in the liver.

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[4] See Hospital Respiratory Protection Program Toolkit, May 2015 at https://www.osha.gov/sites/default/files/publications/OSHA3767.pdf

[5] See OSHA Section 1910.134 Respiratory Protection at https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.134

[6] See OSHA Bloodborne pathogens – Section 1910.1030 - https://www.osha.gov/laws-regs/regulations/standardnumber/1910/1910.1030

h. In general, no vaccine, whether the hepatitis B vaccine or a Covid-19 vaccine, cures or eliminate a communicable diseases 100%.

i. While the main purpose of New York City Department of Health Covid Emergency Orders is to reduce the spread of Covid-19 in the workplace of New York City facilities, the Emergency Orders also carry the unintended consequence of introducing "new hazards" into the body of City employees via the Covid vaccines that can directly affect the health and safety of the City's employees which conflicts with OSHA.

j. The new hazard(s) include the known and reported severe and life-threatening adverse effects from the injection of the Covid-19 vaccine. All healthcare administrators of vaccines are required to report adverse effects of any vaccine to the Centers for Disease Control and Prevention (CDC) Vaccine Adverse Events Reporting System. As of March 18, the system reported that between December 14, 2020, and March 11, 2022, 1,183,495 reports of adverse events from all age groups following COVID vaccines, including 25,641 deaths and 208,209 serious injuries have been reported. As of the dates of the NYC and NYS Covid Emergency Orders were issued, in the VAERS data released September 17, 2021, by the CDC showed a total of 701,561 reports of adverse events from all age groups following COVID vaccines, including 14,925 deaths and 91,523 serious injuries between Dec. 14, 2020 and Sept. 10, 2021.[7]

k. Because the OSHA General Duty Clause at 29 U.S.C. §654[8] requires employers to recognize hazards that are "likely to cause death or serious physical harm to …employees" and to comply with the OSHA standards promulgated to eliminate or reduce a hazard, when evaluated comprehensively, the OSH Act does not list vaccines

[7] See VAERS Reporting Requirements for Covid-19 Vaccines at https://vaers.hhs.gov/reportevent.html
[8] See OSH Act of 1970 Genera Duty Clause 29 U.S.C. 654 at https://www.osha.gov/laws-regs/oshact/section5-duties

as a promulgated standard that eliminates or reduces occupational environmental airborne contaminates or atmospheric contaminants in a workplace.[9]

i. Finally, OSHA standards allow employers to modify work locations also to eliminate an employee's exposure to hazards in the workplace. Remote work is effective in eliminating employee exposures to airborne contaminates that may be in a workplace and is a required to be used by employers before the use of other methods that introduce hazards like vaccines.

19. I am not aware of employees having been terminated for refusing a Hep B vaccine after exposure, therefore there is not need to terminate an employee for refusing to submit to the Covid-19 vaccine.

Additional Opinions Regarding Other Workplace Safety Duties Related to Covid-19

20. According to a CDC report around November 2020[10] before Covid vaccines became available in the U.S., the primary cause of a person suffering severe Covid or a Covid related death after exposure to the respiratory hazard is the existing of one or more pre-existing chronic disease like heart disease, diabetes, chronic livers disease, chronic pulmonary disease, to name a few.

21. The CDC for years has identified poor diet as one of four causes of chronic disease[11] in the U.S., which are the leading causes of all death.[12]

22. For many years, scientific medical journals have concluded that the consumption of red meat and processed meat are the leading cause of most chronic disease and death in the United States.[13]

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[9] See OSH Act of 1970 Comprehensive Table of OSHA laws & Regulations - https://www.osha.gov/laws-regs/regulations/standardnumber

[10] Centers for Disease Control and Prevention (CDC). Coronavirus disease 2019 (COVID-19)—people with certain medical conditions. Atlanta (GA): US Department of Health and Human Services, CDC; Nov. 2020. https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/people-with-medical-conditions.html

[11] Centers for Disease Control and Prevention (CDC), Publication by the National Center for chronic Disease Prevention and Health Promotion – "About Chronic Disease" https://www.cdc.gov/chronicdisease/about/index.htm

[12] National, Heart, Lung and Blood Institute - publication "Americans poor diet drives $50 billion a year in health care costs December 17, 2019" https://www.nhlbi.nih.gov/news/2019/americans-poor-diet-drives-50-billion-year-health-care-costs

[13] "Red meat and processed meat consumption and all-cause mortality:" a meta-analysis

23. New York law defines "potentially hazardous food" as any food that consists in whole or in part of milk or milk products, eggs, meat, poultry, fish, shellfish, edible crustacea, cooked potato, in a form capable of supporting: (1) rapid and progressive growth of infectious or toxigenic microorganisms; or (2) the slower growth of C. botulinum.[14]

24. While the NY State and FDA defines potentially hazardous foods based on the ability of the "food" to support or serve as reservoirs of harmful and infectious pathogens, which include pathogenic protozoans, bacteria, and viruses, as a public health researcher and practitioner, it is my opinion that potentially hazardous foods also include animal foods whose intrinsic factors (which include but are not limited to animal blood, fat and flesh) when consumed have demonstrated in over a dozen scientific studies to cause chronic disease and impairment of the body's natural immune response.

25. Base on my medical experience and knowledge as a medical practitioner who prescribes (as a scientifically supported evidence based intervention) whole plant-based foods and lifestyle interventions to treat chronic disease, including heart disease, renal disease, obesity, both in the clinical and acute and intensive care setting, it is my opinion that employers that provide employees food or meals in the workplace also have a duty to remove and eliminate "potentially hazardous food" from employer operated or contracted cafeterias and specifically from patient meal services and vending machines to also reduce the risk of employees and patients suffering severe Covid or Covid related illnesses.

26. In a study published June 11, 2018 by the CDC that included 5,222 employees across the US, it was found that the foods people get at work tended to be high in empty calories —

Susanna C Larsson, Nicola Orsini, Am J Epidemiol Feb. 1, 2014;179(3):282-9. doi: 10.1093 https://pubmed.ncbi.nlm.nih.gov/24148709/ see also "The global diabetes epidemic as a consequence of lifestyle-induced low-grade inflammation" by H. Kolb and T. Mandrup-Poulsen, Diabetologia Jan, 2010;53(1):10-20. - https://pubmed.ncbi.nlm.nih.gov/19890624/

[14] See New York Codes, Rules and Regulations Section 14-2.3.

those from solid fats and/or added sugars — with more than 70 percent of the calories coming from food that was obtained for free in the workplace.[15]

27. In a 2019 scientific study by a Dr. Robert Vogel (which was summarized in the documentary The Game Changers,[16]) on the impact of the daily consumption of animal fat on human endothelial function, it was determine that the consumption of a single meal that consists of "potentially hazardous food" impairs blood flow throughout the body.

28. Many studies have shown that impaired endothelial function has a direct impact on immune function that can cause severe disease and death.

29. In a study published in April 2021, before any Covid-19 mandates were order, it was reported that endothelial dysfunction and immunothrombosis as key pathogenic mechanisms in severe COVID-19 and Covid related deaths.[17]

30. Therefore, while implementing the most~~potentially~~ effective risk mitigation control to remove the existence of Covid viral pathogens from the workplace atmosphere either through: 1) HEPA filtration systems, 2) ~~reducing an employee's risk of exposure~~ through ~~the use of~~ remote work, or 3) through the use of PAPR respirators to eliminate an employees exposure to the airborne pathogen (either singularly or in combination), in my opinion, removing the "potentially hazardous foods" is equally necessary, if not more important to preventing severe Covid-19 and death in employees.

31. The statements and opinions made in this Affidavit are preliminary and I reserve the right to add to, amend or modify my opinions as more facts are provided during the course of any litigation of the claims by the Classes of Plaintiffs for which this affidavit is provided.

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[15] Foods and Beverages Obtained at Worksites in the United States by Stephen Onufrak CDC Epidemiologist, in Journal of the American Academy of Nutrition and Dietetics 119(6) DOI:10.1016/j.jand.2018.11.011

[16] 3 Minute video on the Impact on Animal Fat on Endothelial Function study by Dr. Robert Vogal, Cardiologist– 2019 study from the "Game Changers" documentary https://tinyurl.com/5du5nuke

[17] Endothelial dysfunction and Immunothrombosis as key pathogenic mechanisms in COVID-19 By Aldo Bonaventura, and Alessandra Vecchié…. Nat Rev. Immunol. 2021; 21(5): 319–329 – see https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8023349/

I declare under penalty of perjury under the laws of the State of Texas that the foregoing is true and correct.

Dated this 19th day of April, 2022.

DR. BAXTER MONTGOMERY MD

A NOTARY PUBLIC OR OTHER OFFICER COMPLETING THIS CERTIFICATE VERIFIES ONLY THE IDENTITY OF THE INDIVIDUAL WHO SIGNED THE DOCUMENT TO WHICH THIS CERTIFICATE IS ATTACHED, AND NOT THE TRUTHFULNESS, ACCURACY, OR VALIDITY OF THAT DOCUMENT.

Subscribed and sworn to (or affirmed) before me on this 19th day of April, 2022, by Dr. Baxter Montogery, proved to me on the basis of satisfactory evidence to be the person(s) who appeared before me.

Witness my hand and official seal.

NANCY NAVA
Notary Public, State of Texas
Comm. Expires 05-25-2025
Notary ID 133120530

Signature of Notary Public

[Affix Notary Seal]

1. The California Respirator Program Administrators toolkit can be accessed at: https://www.cdph.ca.gov/Programs/CCDPHP/DEODC/OHB/Pages/RespToolkit.aspxexternal icon
2. Beckman S, Materna B, Goldmacher S, Zipprich J, D'Alessandro M, Novak D, Harrison R [2013]. Evaluation of respiratory protection programs and practices in

BAXTER DELWORTH MONTGOMERY, MD

The Plant-Based Physician
Montgomery Heart & Wellness
Video Bio

EXPERIENCE:

Clinical Assistant Professor
The University of Texas Health Science Center
Department of Medicine
Division of Cardiology/Clinical Cardiac Electrophysiology

President and CEO
Houston Associates of Cardiovascular Medicine, PA.
(1997-Present)

Executive Director
The Johnsie and Aubary Montgomery Institute of Medical Education and Research (a 501(c) 3 nonprofit organization)

BIRTHPLACE:

Houston, Texas
United States of America

OFFICE ADDRESS:

10480 South Main Street
Houston, Texas 77025
(713) 599-1144 phone
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bmontgomery@drbaxtermontgomery. com

UNDERGRADUATE EDUCATION:

William Marsh Rice University
Houston, Texas
Bachelor's Degree in Biochemistry (1986)

GRADUATE EDUCATION:

The University of Texas Medical Branch at Galveston
Galveston, Texas
Doctor or Medicine

RESIDENCY:

Baylor College of Medicine
Houston, Texas
Internal Medicine

FELLOWSHIP:

The University of Texas Health Science Center at Houston
Houston, Texas
Cardiovascular Diseases
Clinical Cardiac Electrophysiology

CERTIFICATION:

Diplomate of the American Board of Internal Medicine, Cardiovascular Diseases

Diplomate of the American Board of Internal Medicine, Clinical Cardiac Electrophysiology

LICENSURE:

Texas State Board of Medical Examiners (Since 1999)
Permit Number H9549

HOSPITAL APPOINTMENTS:

Attending Physician
Memorial Hermann Hospital - The Texas Medical Center
Houston, Texas

Attending Physician
The Heart and vascular Institute
Memorial Hermann Hospital - The Texas Medical Center
Houston, Texas

Consulting Physician
Select Specialty Hospital - Heights
Houston, Texas

TEACHING RESPONSIBILITES:

Teaching Faculty for Cardiology Fellows and Clinical Advanced Nurse Practitioners
The Heart and Vascular Institute
Memorial Hermann Hospital - The Texas Medical Center
1997 - Present

Cardiovascular Disease Lecturer
GlaxoSmithKline, Inc.
2000 - Present

Cardiovascular Disease Lecturer
Novartis, Inc.
2006 - Present

Cardiovascular Disease Lecturer
Boston Scientific, Inc.
2006 - Present

Co-Director and Lecturing Faculty
Cardiology Concepts for Non-Cardiologists
(An Annual Houston Area Educational Symposium)

JAM Institute, Inc.
2006 - 2008

Steering Committee Member and Lecturing Faculty
Close the Gap
Boston Scientific, Inc.
2006 - Present

RESEARCH:

CLINICAL STUDIES:

ALLHAT: Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial. ALLHAT ALLHAT was a blinded, randomized trial that investigated the relative efficacy of different classes of antihypertensive agents in reducing stroke, illness and death from cardiovascular diseases. A subgroup of patients with hyperlipidemia was randomized comparing Pravastatin compared to usual care.
A Houston Site - Principal Investigator (1998)

INVEST: The International Verapamil SR/Trandolapril Study. INVEST was a randomized controlled clinical trial comparing a calcium antagonist treatment strategy (Isoptin® SR) with a non calcium antagonist treatment strategy for the control of hypertension in a primary care coronary artery disease patient population.
A Houston Site - Principal Investigator (2000)

INVEST SUB-STUDY: This study was a sub-study of the INVEST patient population designed to evaluate the impact of genetic differences on pharmacokinetics.
A Houston Site - Principal Investigator (2000)

The Safety and Efficacy of PNU-182716 Versus Rosiglitazone: This was a one-year, randomized, double blind, parallel group, and active comparator study.
A Houston Site - Principal Investigator (2000)

FACTOR: Fenofibrate and Cerivastatin Trial Optimizing Response. FACTOR was a multicenter, randomized, double blind, placebo controlled, parallel group, study of the safety and efficacy of Cerivastatin in combination with Fenofibrate compared to Cerivastatin alone, Fenofibrate alone and placebo in a population of Type 2 Diabetic Men and Women.
Grant Sponsor - Bayer 2001
A Houston Site - Principal Investigator

ADHERE: ADHERE was a national registry of patients admitted to hospitals with acute decompensated congestive heart failure.
A Houston Site - Principal Investigator (2001)

STELID TM AND STELIX TM LEADS STUDY: This study was a
safety and efficacy study of steroid-eluting cardiac pacing leads.
Grant Sponsor - Ella Medical 2002

ARRHYTHMIA PATHWAY STUDY: This was a patient registry study designed to assess the efficacy of a clinical algorithm for identifying and assessing patients at risk of sudden cardiac arrest.
Grant Sponsor - Medtronic, Inc. 2002
A Houston Site - Principal Investigator

RAPIDO CATHETER STUDY: This study was to evaluate the efficacy of a left ventricular defibrillator-pacemaker lead delivery system.
Grant Sponsor - Guidant, Inc. 2003
A Houston Site - Principal Investigator

PROTOS HEART RATE DISTRIBUTION STUDY: This was a clinical study designed to compare the heart rate distribution in patients undergoing pacemaker implants requiring heart rate response therapy. This study compared the heart rate distribution of accelerometer rate response therapy to the BIOTRONIK Closed Loop System therapy.
Grant Sponsor - Biotronik, Inc. 2003
A Houston Site - Principal Investigator

CSPP100A2404 - A 54 week, randomized, double-blind, parallel-group, multicenter study evaluating the long-term gastrointestinal (GI) safety and tolerability of Aliskiren (300 mg) compared to Ramipril (10 mg) in patients with essential hypertension.
Sponsored by Novartis, since April 4, 2008.
A Houston Site - Principal Investigator

CSPP100AUS03 - An 8 week Prospective, Multicenter, Randomized, Double-Blind, Active Control, Parallel Group Study to Evaluate the Efficacy and Safety of Aliskiren HCTZ versus Amlodipine in African American Patients with Stage 2 Hypertension.
Sponsored by Novartis, since August 2008.
A Houston Site - Principal Investigator

CSPP100A2409- An 8 week randomized, double-blind, parallel-group, multicenter, active-controlled dose escalation study to evaluate the

efficacy and safety of Aliskiren HCTZ (300/25 MG) compared to Amlodipine (10 mg) in patients with satage 2 systolic hypertension and diabetes mellitus.
Sponsored by Novartis, since December 2008.
A Houston Site - Principal Investigator

SPAlOOAUSOl - An 8 week randomized, double-blinded, parallel-group, multicenter, active-controlled dose escalation study to evaluate the efficacy and safety of Aliskiren Administered in Combination with Amlodipine (150/5 mg, 300/10 mg) versus Amlodipine alone (5 mg, 10 mg) in African American patient with Stage 2 Hypertension.
Sponsored by Novartis, since February 2009.

CLAF237B22Ol- A multicenter, randomized, double-blind study to evaluate the efficacy and long-term safety of vildagliptin modifies release (MR) as monotherapy in patients with type 2 diabetes.
Sponsored by Novartis, since February 2009.
A Houston Site - Principal Investigator

CLAF237B2224 - A multi-center, randomized, double-blind study to evaluate the efficacy and long-term safety of vildagliptin modified release (MR) as add-on therapy to metformin in patients with type 2 diabetes.
Sponsored by Novartis, since February 2009.
A Houston Site - Principal Investigator

Galaxy study: An aftermarket registry of one of the Biotronik implantable cardioverter defibrillators ICD leads (2009 to present)
A Houston Site - Principal Investigator

Paradigm study: A multicenter, randomized, double-blind, parallel group, active-controlled study to evaluate the efficacy and safety of LCZ696 compared to enalapril on morbidity and mortality in patients with chronic heart failure and reduced ejection fraction. 2009 -2014
A Houston Site - Principal Investigator

BASIC RESEARCH:

In Rapid Separation of Mitochondria from Extra- mitochondrial Space Applied to Rat Heart Mitochondria. An abstract presented at an NIH sponsored student research poster session, Univ. of Texas Medical Branch, Galveston, TX, June 17, 1987.

Regulation of the Adenine Nucleotide Pool-Size of Heart Mitochondria by the ADP/ATP Translocase. Abstract and poster presented at the Galveston-Houston Conference for Cardiovascular

Research, Univ. of Texas, Medical Branch, Galveston, TX, February 26, 1988.

The Adenine Nucleotide Pool-Size of Heart Mitochondria is Regulated by the ADP/ATP Translocase. Abstract presented at the 29th Annual National Student Research Forum, University of Texas Medical Branch, Galveston Texas, April 6-8, 1988.

Increased Frequency of the Deletion Allele of the ACE Gene in African-Americans Compared to Caucasians. This study evaluated the prevalence of the deletion allele of the ACE gene in a population of African Americans compared to Caucasians. The findings were presented at the annual meeting of the American College of Cardiology in March of 1996.

Determination of the effect of Calcium infusion on CGRP mRNA Production. A pilot study investigating a possible mechanism by which calcium supplementation may increase CGRP (Calcitonin gene-related peptide, a potent peripheral vasodilator) content in afferent neurons of Sprague Dawley rats, 1990.

PUBLICATIONS:

Montgomery, B, D, MD. A Review of Microanatomy for Medical Students, 1987, chapter 1-8.

Baxter D. Montgomery, MD, Elizabeth A. Putnam, Ph.D., John Reveille, MD, Dianna M. Milewicz. MD, Ph.D.: Increased Frequency of the Deletion Allele of the ACE Gene in African-Americans Compared to Caucasians. (Abstract) J. American College of Cardiology March, 1996

Doyle, N.M., Monga, M., **Montgomery, B.**, Dougherty, A.H.: Arrhythmogenic right ventricular cardiomyopathy with implantable cardioverter defibrillator placement in pregnancy. J Mat Fetal Neo Med 18:141-4, 2005

Baxter D. Montgomery, MD Co-Author of Dreams of the nation Book: "Improving Health" with focus on strengthening the food and health connection and replacing unnatural foods from our diet and replacing them with natural foods as a way of reversing illness. 2009

Montgomery, Baxter D: The Food Prescription for Better Health, Houston: Delworth Publishing, 2011

Montgomery,B.D, MD, Effects of the Montgomery Food Prescription on Clinical Biomarkers of Cardiovascular Disease. Plant-based diet can improve clinical biomarkers associated with cardiovascular disease. This study was submitted to the 10th annual Texas A&M University System Pathways Student Research Symposium 2012.

Baxter D. Montgomery, MD Co-Author of the book Rethink Food: About the need for revolutionary change in how to address chronic illness with optimal nutrition.2014

CLINICAL PRESENTATIONS:

Clinical Concepts for Non Cardiologist, Director and Faculty. An educational symposium held for primary care and other non-cardiology specialists in the Houston area. October 2006

Patients at Risk for Sudden Cardiac Arrest Dinner Symposium at the Houston Forum June, 2007

Clinical Concepts for Non Cardiologist, Director and Faculty. An educational symposium held for primary care and other non-cardiology specialists in the Houston area. October 2007

Clinical Concepts for Non Cardiologist, Director and Faculty. An educational symposium held for primary care and other non-cardiology specialists in the Houston area. October 2008

Houston Town Hall Meeting, Director and Faculty. Health summit on the benefits of a healthy nutritional lifestyle for the management of chronic illnesses held for both health care professional and the general public in the Houston area. 2009

Houston Town Hall Meeting, Director and Faculty. Health summit on the benefits of a healthy nutritional lifestyle for the management of chronic illnesses held for both health care professional and the general public in the Houston area. 2010

Houston Health Summit (Town Hall Meeting), Director and Faculty. Health summit on the benefits of a healthy nutritional lifestyle for the management of chronic illnesses held for both health care professional and the general public in the Houston area. 2011

Houston Health Summit (Town Hall Meeting), Director and Faculty. Health summit on the benefits of a healthy nutritional lifestyle for the management of chronic illnesses held for both health care professional and the general public in the Houston area. 2012

Houston Health Summit (Town Hall Meeting), Director and Faculty. Health summit on the benefits of a healthy nutritional lifestyle for the management of chronic illnesses held for both health care professional and the general public in the Houston area. 2013

PROFESSIONAL APPOINTMENTS:

Clinical Assistant Professor of Medicine, University of Texas Health Science Center - Houston 1996 - Present

Steering Committee Member, Boston Scientific Close the Gap Initiative 2005 - Present
Scientific/Medical Board of Advisors, Nutritional Excellence, Inc. 2007 - Present

Medical Board of Directors, Twelve Oaks Medical Center Independent Physician's Association 2005 - Present

Medical Executive Committee (Twelve Oaks Hospital), Member at Large 2002 - 2006

Patient Safety Committee (Twelve Oaks Hospital), Chairman 2002 - 2004

Physician Peer Review Committee (Twelve Oaks Hospital) 2002 - 2005

Medical Director, SCCI (Specialized Complex Care) Hospital, 2003 - 2005

Physician Relation Council Advisory Board, Unicare, 2002 - 2004

Aldine Education Foundation: The mission of the Aldine Education Foundation is to provide community-based support to the Aldine Independent School District in pursuit of excellence in teaching, innovation in the classroom and superior learning opportunities for all students.

CLINICAL INTERESTS:

Nutritional Lifestyle Interventions for the Management of Chronic Illnesses
Cardiac Pacing and Electrophysiology

Diastolic and Systolic Heart Failure
Hypertensive Heart Disease
Cardiovascular Exercise Physiology
Basic Echocardiography
Nuclear Cardiology
Diagnostic Cardiac Catheterization
Cardiovascular Wellness and Nutrition

PROFESSIONAL ASSOCIATIONS:

American College of Cardiology (Elected as Fellow of the College in January, 1999)
American Heart Association
Heart Rhythm Society (North American Society of Pacing and Electrophysiology, NASPE)
American College of Physicians
Harris County Medical Society
Houston Medical Forum

HONORS AND AWARDS:

Benjamin Spock Award for Compassion in Medicine - 2010

America's Top Physicians - 2007

Cumulative evaluation of "Superior" performance by senior house staff and faculty during first year of residency (Baylor College of Medicine), 1990

Outstanding Young Men of America, 1988

Kempner Award (University of TX Medical Branch) 1986-87 and 1987-88

Academic Scholarship (University of TX Medical Branch) 1986-87

Who's Who Among American Colleges and Universities (Rice University) 1986

Franz Brotzen Outstanding Senior Award (Rice University) 1986

Jones College Service Award (Rice University) 1986 and 1985

100 Black Men of Metropolitan Houston (Awarded in 2012) for the dedication to the improvement of the community.

Physicians Committee for Responsible Medicine- Member of Advisory Board- Current.

ACTIVITIES:

Gardening
Scouting
Physical Conditioning

Received: 1 June 2018 | Revised: 7 July 2018 | Accepted: 12 July 2018

DOI: 10.1002/clc.23027

WILEY CLINICAL CARDIOLOGY

CLINICAL INVESTIGATIONS

Consumption of a defined, plant-based diet reduces lipoprotein(a), inflammation, and other atherogenic lipoproteins and particles within 4 weeks

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Background: Lipoprotein(a) [Lp(a)] is a highly atherogenic lipoprotein and is minimally effected by lifestyle changes. While some drugs can reduce Lp(a), diet has not consistently shown definitive reduction of this biomarker. The effect of consuming a plant-based diet on serum Lp(a) concentrations have not been previously evaluated.

Hypothesis: Consumption of a defined, plant-based for 4 weeks reduces Lp(a).

Methods: Secondary analysis of a previous trial was conducted, in which overweight and obese individuals (n = 31) with low-density lipoprotein cholesterol concentrations >100 mg/dL consumed a defined, plant-based diet for 4 weeks. Baseline and 4-week labs were collected. Data were analyzed using a paired samples t-test.

Results: Significant reductions were observed for serum Lp(a) (−32.0 ± 52.3 nmol/L, P = 0.003), apolipoprotein B (−13.2 ± 18.3 mg/dL, P < 0.0005), low-density lipoprotein (LDL) particles (−304.8 ± 363.0 nmol/L, P < 0.0005) and small-dense LDL cholesterol (−10.0 ± 9.2 mg/dL, P < 0.0005). Additionally, serum interleukin-6 (IL-6), total white blood cells, lipoprotein-associated phospholipase A2 (Lp-PLA2), high-sensitivity c-reactive protein (*hs*-CRP), and fibrinogen were significantly reduced ($P \leq 0.004$).

Conclusions: A defined, plant-based diet has a favorable impact on Lp(a), inflammatory indicators, and other atherogenic lipoproteins and particles. Lp(a) concentration was previously thought to be only minimally altered by dietary interventions. In this protocol however, a defined plant-based diet was shown to substantially reduce this biomarker. Further investigation is required to elucidate the specific mechanisms that contribute to the reductions in Lp(a) concentrations, which may include alterations in gene expression.

KEYWORDS

general clinical cardiology/adult, lipoproteins, preventive cardiology, vegetarian diet

1 | INTRODUCTION

Lipoprotein(a) [Lp(a)] is an atherogenic lipoprotein structurally similar to low-density lipoprotein cholesterol (LDL-C), although synthesis occurs through independent pathways. Key differences include the linkage of apolipoprotein B100 (Apo-B) to apolipoprotein(a) on the LDL surface.[1,2] It has been estimated that expression of the genomic region encoding apolipoprotein(a) (*LPA* gene) accounts for approximately 90% of plasma Lp(a) concentrations.[3] Elevated Lp(a) is independently associated with cardiovascular disease,[4] and the LPA gene was observed to have the strongest genetic link to cardiovascular disease.[5] Individuals with Lp(a) plasma concentrations >20 mg/dL have twice the risk of developing cardiovascular disease and approximately 25% of the population may have this plasma concentration.[6] The mode of action by which Lp(a) exerts its atherogenic effect is likely similar to that of LDL-C, by deposition in the sub-endothelial space and uptake by macrophages mediated via the VLDL receptor.[7] Lp(a) is particularly atherogenic due to its unique property of being a carrier of oxidized phospholipids, in addition to its higher binding affinity to negatively charged endothelial proteoglycans.[8] Lp(a) can facilitate

endothelial dysfunction when concentrations are elevated likely due to this effect.[9]

While PCSK9 inhibitors, high dose atorvastatin, ezetimibe and niacin have resulted in significant reductions in Lp(a),[10–12] lifestyle interventions have not reliably demonstrated reduced Lp(a) to a clinically significant degree. Interestingly, even high saturated fat and high cholesterol diets known to induce hypercholesterolemia have had little influence on plasma Lp(a) concentrations.[13] Despite the lack of evidence in the literature indicating a relationship between diet and Lp(a) concentrations, a defined, plant-based has not been previously evaluated with respect to its potential effect to reduce Lp(a). Previous investigations have found that a very-high fiber diet comprised of vegetables, fruits and nuts can reduce LDL-C by 33% and Apo-B by 26%,[14] although Lp(a) was not measured. Since such a diet can result in dramatic reductions in LDL-C and Apo-B, secondary analysis of a previously published investigation[15] employing a similar plant-based diet were analyzed to evaluate if Lp(a) could be significantly reduced after 4 weeks among other inflammatory indicators and atherogenic lipoproteins and particles.

2 | METHODS

2.1 | Study population

Participants were subjects of a previous study in which written informed consent was obtained to draw blood for analysis.[15] Laboratory reports for each subject included biomarkers used for clinical purposes, and selected biomarkers are included in the present investigation. The study protocol was approved by the Texas Woman's University Institutional Review Board, Houston.

The study protocol has been previously described.[15] Briefly, all participants were registered new patients of a cardiovascular center and were hypertensive (systolic blood pressure ≥ 140 mmHg or diastolic blood pressure ≥ 90 mmHg), had elevated LDL-C (≥100 mg/dL) and excess body weight (body mass index ≥25 kg/m^2) at baseline. Exclusionary criteria included current tobacco use, current drug abuse, excessive alcohol use (>2 glasses of wine or equivalent for men or > 1 glass of wine or equivalent for woman), a current cancer diagnosis, an ongoing clinically defined infection, a mental disability that would prevent a participant from following the study protocol, an estimated glomerular filtration rate < 60 mg/dL, current pregnancy or lactation, a hospitalization within the past 6 months, and previous exposure to the nutrition program.

2.2 | Intervention

Participants were instructed to consume a defined, plant-based diet for 4 weeks ad-libitum which included the consumption of foods within a food classification system.[15] These foods fell within food levels 0 to 4b of the food classification system (Table S1, Supporting information). Briefly, excluded were animal products, cooked foods, free oils, soda, alcohol, and coffee. Allowed for consumption were raw fruits, vegetables, seeds, and avocado. Small amounts of raw buckwheat and oats were also permitted. Vitamin, herbal, and mineral supplements were to be discontinued unless otherwise clinically indicated. All meals and snacks were provided to subjects, although they were free to consume food on their own within food levels 0 to 4b. In addition, subjects were not advised to alter their exercise habits. Adherence was measured daily as previously described[15] with an adherence assessment tool. Participants indicated in writing each day whether they were adherent. Dietary recalls (24-hour) were conducted by a trained nutritionist at baseline and at 4 weeks. Nutrient intake was analyzed by the Nutrition Data System for Research software (University of Minnesota, version 2016). No lipid lowering medications were altered throughout the intervention.

2.3 | Measures

After a 12-hour fast, the following plasma biomarkers were obtained at baseline and after 4-weeks: total cholesterol (Total-C), LDL-C, high-density lipoprotein cholesterol (HDL-C), triglycerides, LDL particles (LDL-P), small-dense low-density lipoprotein cholesterol (sdLDL-C), Apo-B, high-density lipoprotein 2 cholesterol (HDL2-C), apolipoprotein A-1 (Apo A-1), and Lp(a). Additionally, high-sensitivity c-reactive protein (*hs*-CRP), endothelin, interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-a), lipoprotein-associated phospholipase A2 (Lp-PLA2), myeloperoxidase, fibrinogen, troponin-I, N-terminal pro b-type natriuretic peptide (NT-proBNP), total white blood cell count (WBC), neutrophil count, lymphocyte count, monocyte count, eosinophil count, and basophil count were documented. These specific biomarkers of interest were analyzed by either True Health Diagnostics (Frisco, Texas) or Singulex (Alameda, California) depending on the subject's health insurance. The same company that analyzed the baseline labs for a participant was used for the follow-up labs to ensure consistency.

2.4 | Data analysis

Paired samples t-tests were used for the analysis of biochemical measures at baseline and 4-weeks, and significance was confirmed with non-parametric tests. Significance was determined to be a P value less than 0.05. SPSS (version 24) was used for data analysis.

3 | RESULTS

Baseline demographics are indicated in Table 1. Subjects represent a sample that was 81% obese with multiple clinical diagnoses. Two-thirds of subjects were women and 80% were African American.

Adherence to the dietary intervention was approximately 87% over the course of the 4 weeks as measured by the daily adherence assessment tool. Food group consumption is indicated in Table 2 at baseline and 4-weeks. Notably, total fruit consumption increased from 1.3 ± 2.0 servings to 11.8 ± 10.4 servings (808% increase, $P < 0.0005$) and total vegetable consumption increased 2.7 ± 2.0 servings to 16.0 ± 9.2 servings (493% increase, $P < 0.0005$). Additionally, total animal product consumption decreased from 7.9 ± 4.7 servings to 0.4 ± 1.4 servings (95% decrease, $P = 0.001$). The consumption of avocados, dark-green vegetables, deep-yellow vegetables, tomatoes,

TABLE 1 Baseline characteristics and clinical diagnoses

	Participants[a]
n	31
Age (years)	53.4 (32-69)
Sex	
Male	10 (33%)
Female	21 (67%)
Race, ethnicity	
African American	25 (80%)
Hispanic	3 (10%)
White	3 (10%)
Mean BMI (kg/m^2)	37.5 ± 8.3
Overweight (25-29.9 kg/m^2)	6 (19%)
Obesity class 1 (30-34.9 kg/m^2)	6 (19%)
Obesity class 2 (35-39.9 kg/m^2)	10 (33%)
Obesity class 3 (≥40 kg/m^2)	9 (29%)
Current diagnoses	
Coronary artery disease	10 (33%)
Type II diabetes mellitus	8 (27%)
Arthritic condition	7 (23%)
Pre-diabetes	5 (17%)

Abbreviation: BMI, body mass index.
[a] Data are mean (range) unless otherwise indicated.

and other vegetables also significantly increased ($P \leq 0.006$). A decreased consumption of white potatoes, fried potatoes, total grains, refined grains, whole grains, added oils, added animal fat, red meat, white meat, eggs, and dairy were also observed ($P \leq 0.027$). The consumption of sweets (5% decrease, $P = 0.90$) and the consumption of nuts/seeds (17% increase, $P = 0.736$) did not significantly change between baseline and 4-weeks.

Body weight, BMI, total cholesterol, LDL-C, HDL-C, and triglycerides (Table 3) were significantly reduced after 4-weeks of the dietary intervention ($P \leq 0.008$). Lp(a) was also significantly reduced (−32.0 ± 52.3 nmol/L, $P = 0.003$). In addition, LDL-P, sdLDL-C, Apo-B, HDL2-C, and Apo A-1 were significantly reduced ($P \leq 0.03$). Of the atherogenic lipoproteins, sdLDL-C had the greatest relative reduction of approximately 30% (Figure 1). Lp(a) reduced 16% which was proportional to the decrease in Total-C, triglycerides and LDL-P.

Of the inflammatory indicators, *hs*-CRP, IL-6, Lp-PLA2, and fibrinogen significantly decreased ($P \leq 0.004$) (Table 4). The WBC, neutrophil, lymphocyte, monocyte, eosinophil and basophil count also significantly decreased ($P \leq 0.033$). Interestingly, no statistically significant changes were observed for endothelin-1, TNF-a, myeloperoxidase, troponin-I, or NT-proBNP ($P \geq 0.056$) between baseline and 4-weeks.

TABLE 2 Number of food group servings at baseline and 4-weeks[a]

Food group	Serving size	Baseline[b]	Final[b]	Change[c]	*P*[d]
Fruits, total	1/2 cup chopped, 1/4 cup dried or 1 medium piece	1.3 ± 2.0	11.8 ± 10.4	808% (10.5 ± 10.8)	<0.0005
Avocado	1/2 cup chopped	0.1 ± 0.2	0.9 ± 0.9	800% (0.8 ± 0.9)	<0.0005
Vegetables, Total	1/2 cup chopped or 1 cup raw leafy	2.7 ± 2.0	16.0 ± 9.2	493% (13.3 ± 9.2)	<0.0005
Dark-green vegetables	1/2 cup chopped or 1 cup raw leafy	0.7 ± 1	5.2 ± 3.8	643% (4.5 ± 4.0)	<0.0005
Deep-yellow vegetables	1/2 cup chopped	0.2 ± 0.4	1.2 ± 1.1	500% (1.0 ± 1.3)	<0.0005
Tomatoes	1/2 cup chopped	0.4 ± 0.5	1.7 ± 2.4	325% (1.3 ± 2.4)	0.006
Other vegetables	1/2 cup chopped	1.4 ± 1.2	7.9 ± 6.6	464% (6.5 ± 6.3)	<0.0005
White Potatoes[e]	1/2 cup chopped or 1 medium baked potato	0.3 ± 0.7	0.0 ± 0.0	−100% (−0.3 ± 0.7)	0.03
Fried potatoes	1/2 cup chopped or 70 g french fries	0.5 ± 0.9	0.1 ± 0.3	−80% (−0.4 ± 0.9)	0.027
Grains, Total	1 slice of bread or halfcup cooked cereal	5.7 ± 3.5	0.7 ± 0.9	−88% (−5.0 ± 3.6)	<0.0005
Refined grains	1 slice of bread or half cup cooked cereal	3.8 ± 2.7	0.2 ± 0.7	−95% (−3.6 ± 3.0)	<0.0005
Whole grains	1 slice of bread or half cup cooked cereal	1.9 ± 2.6	0.5 ± 0.7	−74% (−1.4 ± 2.7)	0.007
Sweets[f]	4 g of sugar, 1 tbsp honey or 2 tbsp syrup	1.8 ± 2.3	1.7 ± 1.5	−5% (−0.1 ± 2.7)	0.90
Nuts/seeds	1/2 oz	1.2 ± 3.0	1.4 ± 1.6	17% (0.2 ± 3.4)	0.736
Added oils	1 tsp	3.2 ± 3.5	0.1 ± 0.2	−97% (−3.1 ± 3.5)	<0.0005
Added animal fat	1 tsp	1.3 ± 2.3	0.0 ± 0.1	−100% (−1.3 ± 2.3)	0.005
Animal products, Total[g]	1 oz	7.9 ± 4.7	0.4 ± 1.4	−95% (−7.5 ± 5.3)	0.001
Red meat	1 oz	2.1 ± 2.9	0.1 ± 0.2	−95% (−2.0 ± 3.0)	<0.0005
White meat	1 oz	3.9 ± 3.7	0.2 ± 1.1	−95% (−3.7 ± 4.1)	<0.0005
Eggs	1 large egg	0.5 ± 0.7	0.0 ± 0.1	−100% (−0.5 ± 0.7)	0.002
Dairy	1 cup of milk/yogurt or 1.5 oz of cheese	1.5 ± 1.6	0.1 ± 0.3	−93% (−1.4 ± 1.7)	<0.0005

[a] Data are for subjects who completed 24-h recalls at both baseline and 4-weeks ($n = 30$).
[b] Data are listed in serving size and are presented as mean ± SD.
[c] Data indicated as % change (mean ± SD).
[d] Paired samples *t*-tests for within-group comparisons of changes from baseline to final values.
[e] Excludes fried potatoes.
[f] Includes honey, candy, or other added sugars.
[g] Excludes added animal fat.

TABLE 3 Atherogenic lipoproteins and particles at baseline and 4-weeks

	Baseline[a]	Final[a]	Change[b]	*P*[c]
Weight (kg)	108.1 ± 28.6	101.4 ± 26.3	−6% (−6.6 ± 3.6)	<0.0005
BMI (kg/m^2)	37.5 ± 8.3	35.2 ± 7.8	−6% (−2.2 ± 1.1)	<0.0005
Total-C (mg/dL)	216.6 ± 34.2	182.7 ± 29.9	−16% (−33.8 ± 25.9)	<0.0005
LDL-C (mg/dL)	143.0 ± 28.9	118.4 ± 26.4	−17% (−24.6 ± 21.3)	<0.0005
HDL-C (mg/dL)	54.8 ± 9.4	49.5 ± 10.6	−9% (−5.2 ± 6.2)	<0.0005
Triglycerides (mg/dL)	124.1 ± 58.1	104.5 ± 53.6	−16% (−19.6 ± 38.4)	0.008
Lp(a) (nmol/L)[d]	200.7 ± 150.0	168.8 ± 126.7	−16% (−32.0 ± 52.3)	0.003
Apo-B (mg/dL)	115.2 ± 24.5	101.9 ± 17.7	−11% (−13.3 ± 18.3)	<0.0005
LDL-P (nmol/L)[e]	1891 ± 586	1586 ± 508	−16% (−305 ± 363)	<0.0005
*sd*LDL-C (mg/dL)	33.7 ± 11.5	23.7 ± 8.7	−30% (−10.0 ± 9.2)	<0.0005
HDL2-C (mg/dL)	17.4 ± 9.8	15.6 ± 9.9	−10% (−1.8 ± 4.5)	0.030
Apo A-1 (mg/dL)	189.7 ± 150.7	160.2 ± 126.5	−14% (−27.0 ± 19.6)	<0.0005

Abbreviations: Apo A-1, apolipoprotein A-1; Apo-B, apolipoprotein B100; BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; HDL2-C, high-density lipoprotein-2 cholesterol; LDL-C, low-density lipoprotein cholesterol; LDL-P, low-density lipoprotein particles; Lp(a), lipoprotein(a); sdLDL-C, small-dense low-density lipoprotein cholesterol; total-C, total cholesterol.
[a] Mean ± SD (*n* = 31 unless otherwise indicated).
[b] Data indicated as % change (mean ± SD).
[c] Paired samples *t*-tests for within-group comparisons of changes from baseline to final values.
[d] *n* = 28 due to premature coagulation of sample (*n* = 1) and incompatible units (mg/dL) when merging laboratory results (*n* = 2).
[e] *n* = 29 due to premature coagulation of samples.

4 | DISCUSSION

The consumption of a defined, plant-based diet resulted in a significant reduction in Lp(a) after 4 weeks; thus, the study hypothesis was accepted. The reduction in Lp(a) was profound and is one of the largest reductions due to lifestyle reported in the literature. The magnitude of change was comparable to other leading medical therapies, such as niacin (~20% reduction) and PCSK9 inhibitors (~25% reduction).[12] It is important to note that this dietary intervention rapidly reduced Lp(a) by 16% in only 4 weeks, whereas shorter duration niacin and PCSK9 inhibitor drug trials typically lasted 8 to 12 weeks. It should also be noted that niacin may reduce inflammation, such as *hs*-CRP, by 15% after 3 months, although PCSK9 inhibitors do not.[16,17] After 4 weeks, the dietary intervention reduced *hs*-CRP by 30.7%. In addition, IL-6, Lp-PLA2, fibrinogen, and white blood cells were significantly reduced, as were sdLDL-C, LDL-P, and Apo-B, all of which represent a systemic, cardio-protective effect.[18–24] Thus, the use of this single dietary approach in the clinical setting, vs multiple drug therapy, may be an appropriate tool in treating complex patients with a myriad of elevated CVD-related biomarkers.

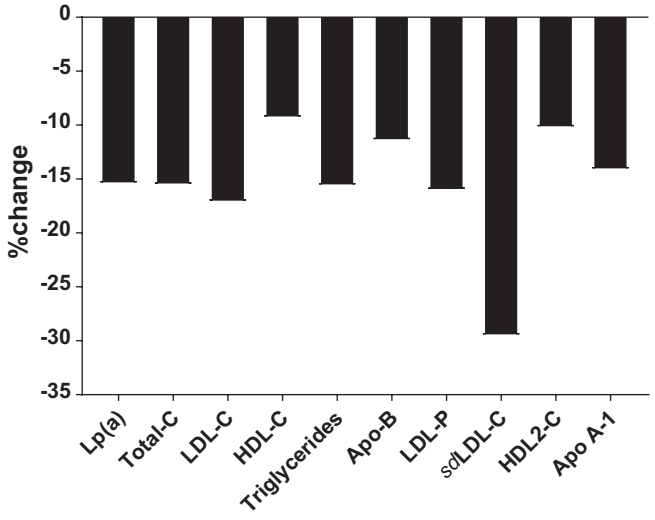

FIGURE 1 Percent change of atherogenic lipoproteins and particles from baseline to 4-weeks. All variable changes indicated are significant ($P < 0.05$). Lp(a), lipoprotein(a); Total-C, total cholesterol; LDL-C, low-density lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; Apo-B, apolipoprotein B100; LDL-P, low-density lipoprotein particles; sdLDL-C, small-dense low-density lipoprotein cholesterol; HDL2-C, high-density lipoprotein-2 cholesterol; Apo A-1, apolipoprotein A-1

Elevated Apo A1, HDL-C, and HDL2-C are associated with reduced cardiovascular disease risk.[24,25] While these HDL fractions were significantly reduced in this trial, this is a common phenomenon observed when consuming plant-based diets. A systematic review and meta-analysis of plant-based observational and clinical trials found that while HDL-C was significantly reduced compared to those consuming non-vegetarian diets, LDL-C and total-C were also reduced.[26] Despite reductions in HDL-C, those who consumed plant-based diets had a 25% reduced incidence of ischemic CVD compared with non-vegetarian counterparts.[27]

Lp(a) concentrations in the present study represent a high-risk population.[28] This may be explained by the higher proportion of African Americans in this sample, as African Americans may have higher Lp(a) concentrations compared with Caucasians.[29] An evaluation of 532 359 patients found that an Lp(a) concentration > 50 mg/dL was common among patients.[30] This range roughly corresponds to the mean nmol/L Lp(a) concentration observed in the present study.

4.1 | Effect of weight loss on plasma Lp(a) concentrations

An energy restricted diet was found to independently reduce serum Lp(a) in those with baseline concentrations >20 mg/dL, but not <20 mg/dL.[31] Further studies have found that weight loss may not

TABLE 4 Inflammatory and other cardiovascular indicators at baseline and 4-weeks

	Baseline[a]	Final[a]	Change[b]	P[c]
hs-CRP (mg/dL)	7.8 ± 6.4	5.4 ± 4.7	−30.7% (−2.4 ± 3.7)	0.001
Endothelin (pg/mL)[d]	2.2 ± 0.7	2.2 ± 0.8	0% (0.0 ± 0.7)	0.916
IL-6 (pg/mL)[d]	2.6 ± 1.4	2.0 ± 1.0	−23.1% (−0.6 ± 1.0)	0.001
TNF-α (pg/mL)[d]	2.0 ± 0.9	2.2 ± 0.9	10.0% (0.2 ± 0.6)	0.096
Lp-PLA_2 (ng/mL)[d]	252.3 ± 136.3	210.7 ± 119.1	−16.4% (−41.6 ± 64.6)	0.001
Myeloperoxidase (pmol/L)[e]	124.1 ± 58.1	104.5 ± 53.6	−23.0% (−28.5 ± 66.1)	0.056
Fibrinogen (mg/dL)[f]	561.4 ± 112.2	530.1 ± 102.9	−5.6% (−31.3 ± 50.7)	0.004
NT-proBNP (pg/mL)[d]	65.2 ± 71.2	69.4 ± 75.9	6.2% (4.1 ± 23.2)	0.337
Total WBC (K/μL)[d]	6.3 ± 2.0	4.8 ± 1.3	−22.2% (−1.4 ± 1.1)	<0.0005
Neutrophils (K/μL)[d]	3.5 ± 1.4	2.5 ± 0.9	−28.6% (−1.0 ± 0.8)	<0.0005
Lymphocytes (K/μL)[d]	1.9 ± 0.7	1.6 ± 0.6	−15.8% (−0.3 ± 0.4)	<0.0005
Monocytes (K/μL)[d]	0.46 ± 0.12	0.38 ± 0.09	−15.2% (−0.07 ± 0.1)	<0.0005
Eosinophils (K/μL)[d]	0.18 ± 0.11	0.15 ± 0.11	−16.6% (−0.03 ± 0.07)	0.033
Basophils (K/μL)[d]	0.029 ± 0.016	0.024 ± 0.015	−17.2% (−0.005 ± 0.010)	0.016

Abbreviations: *hs*-CRP, high-sensitivity c-reactive protein; IL-6, interleukin-6; Lp-PLA_2, lipoprotein-associated phospholipase A2; NT-proBNP, N-terminal pro b-type natriuretic peptide; TNF-α, tumor necrosis factor-alpha; WBC, white blood cells.
[a] Mean ± SD (*n* = 31 unless otherwise indicated).
[b] Data indicated as % change (mean ± SD).
[c] Paired samples *t*-tests for within-group comparisons of changes from baseline to final values.
[d] *n* = 30 due to premature coagulation of samples.
[e] *n* = 25 due to premature coagulation of samples.
[f] *n* = 27 due to premature coagulation of samples.

independently reduce Lp(a) concentrations. A pooled analysis of cohorts found that as weight loss ensued, Lp(a) concentrations surprisingly increased.[32] Baseline Lp(a) concentrations on average between the four cohorts analyzed were approximately 40 mg/dL, well above the >20 mg/dL threshold reported in the initial study.[31] Other investigations examining the effect of weight loss on Lp(a) concentration have not demonstrated a relationship between these two variables.[33,34] Interestingly, the emphasis on consuming plant-based foods, even with a calorie restricted diet, did not result in Lp(a) reductions compared with a calorie restricted red meat centered diet.[35] The plant-centered diet in this trial[35] still contained a significant number of calories derived from animal-based sources in addition to processed plant foods. Also, both diets contained similar quantities of dietary fiber, a measure of plant-food intake. Based on these weight loss trials, Lp(a) concentration is likely not influenced by weight reduction.

4.2 | Effect of diet on plasma Lp(a) concentrations

Other trials using diets emphasizing plant-based foods have not demonstrated similar results. A low-fat and low-saturated fat diet with an increased intake of fruits and vegetables interestingly increased Lp(a) concentrations.[36] Subjects consumed four to five servings of fruits or berries and five to six servings of vegetables daily for 5 weeks and all food was provided. It is important to note that subjects still consumed animal products throughout the intervention[36] which included dairy products and lean meats. The fiber content (40 g vs 51 g in the present study) was not as high as would be expected when consuming a higher quantity of plant-foods, and the number of fruits and vegetables did not meet the levels observed in the present study (11.8 servings of fruits and 16 servings of vegetables). Based on this data, it is probable that exclusively increasing fruit and vegetable intake is not sufficient to elicit reduced Lp(a) concentrations.

It has also been reported that a low-carbohydrate, high-fat diet (45% carbohydrate, 40% fat) may have a favorable impact on Lp(a) concentrations compared with a high-carbohydrate, low-fat diet (65% carbohydrate, 20% fat), although it is unclear as to what precisely was consumed on either of these diets.[37] In addition, the differences were small, as only a 2.17 mg/dL difference was observed between both groups, and baseline Lp(a) concentrations were <20 mg/dL. The Omni Heart Trial also found that replacing calories from carbohydrates and protein with unsaturated fats produced a smaller increase in Lp(a) comparatively, but both diets still elicited increased plasma Lp(a) compared with baseline. The differences between groups were also small at the end of the intervention (<4 mg/dL difference).[38]

In individuals with low baseline Lp(a) concentrations (approximately 5.5 mg/dL), the consumption of copious saturated fat, cholesterol (derived from egg consumption) and polyunsaturated fat did not influence Lp(a) concentrations.[13] Carbohydrate intake was low in this trial as well (39% to 46% carbohydrate as a percent of energy). While fat consumption does not appear to influence serum Lp(a) concentrations in the fasting state, a variety of fats may significantly increase postprandial, transient plasma Lp(a) concentrations over the course of 8 hours.[39] Investigators found that linoleic, oleic, palmitic, and stearic acid all resulted in significant transient increases in Lp(a) concentrations which closely tied to a proportional increase in triacylglycerol concentrations. While saturated fats, stearic acid and palmitic acid, appeared to have the greatest increase in serum Lp(a) compared with oleic acid and linoleic acid, this differing response did not reach statistical significance.

4.3 | Mechanisms contributing to reduced plasma Lp(a)

The observed reduction in Lp(a) in the present study may be due to decreased hepatic synthesis of apolipoprotein(a) and Apo-B. This may be in part due to decreased expression of the LPA gene. Since the LPA gene is almost exclusively expressed in the liver,[40] hepatic influences, including the production of *hs*-CRP and inflammatory cytokines, such as IL-6, may upregulate LPA gene expression.[41] Indeed, those with inflammatory conditions may have increased Lp(a) concentrations compared with healthy controls.[42]

Current data in our plant-based study supports this hypothesis, as reduced *hs*-CRP and IL-6 was observed. In contrast, previous studies utilizing plant-centered diets to reduce Lp(a) were unsuccessful, as animal products were still substantially consumed.[35,36] Animal-based foods, including lean meat, can induce a postprandial inflammatory response, including increased *hs*-CRP and IL-6.[43] Pooled data of those consuming non-vegan, plant-based diets have shown reduced *hs*-CRP and IL-6,[44] although to a lesser extent compared with the present study (*hs*-CRP; −0.55 mg/dL vs −2.42 mg/dL, IL-6; −0.25 pg/mL vs −0.64 pg/mL). The elimination of animal products and processed foods completely on a defined, plant-based diet may be a more prudent dietary strategy to avoid potential fluctuations in inflammation. Thus, the fact that there were only minimally processed plant foods consumed during this dietary intervention may account for the observed reduction in serum Lp(a) concentrations that may be associated with reduced LPA gene expression. Further mechanistic research is needed to confirm this hypothesis.

4.4 | Strengths and limitations

The high dietary adherence and provision of all food to subjects supports the conclusion that the intervention likely fully accounted for the observed biochemical changes among the subjects. Furthermore, the study took place in an outpatient clinical setting with established patients providing a real-world example of a standard clinical practice. This study provides a model for the implementation of this intervention across other medical practices. In contrast, a limitation in the design of this study was the lack of a control group and the small sample size. A larger sample size and a control group would be needed to strengthen a causal relationship.

5 | CONCLUSION

A defined, plant-based diet has a favorable impact on Lp(a) and other atherogenic lipoproteins and particles. Lp(a) concentration was previously thought to be only minimally altered by lifestyle interventions. In this study, however, a defined plant-based diet resulted in a substantial reduction in Lp(a) in only 4 weeks. Further investigations are warranted to elucidate the specific mechanisms that contribute to reduced Lp(a) concentrations, which may include alterations in LPA gene expression mediated via hepatic inflammation.

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Conflict of interest

The authors declare no potential conflicts of interest.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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